Diseases of Geriatric Guinea Pigs and Chinchillas

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KEYWORDS

• Guinea pig • Chinchilla • Geriatric • Scurvy
• Pododermatitis • Malocclusion

In the United States over the last 10 years, interest has been increasing in small mammal pets, including guinea pigs and chinchillas. This trend has resulted in improved diet and husbandry, which in turn has led to increased longevity of many of these pets. The problems and diseases of the older guinea pig and chinchilla differ to some extent from that of the young newly obtained animal, although diet and husbandry still play important roles.

The guinea pig (*Cavia porcellus*), or cavy, is a species of rodent belonging to the family Caviidae and the genus *Cavia*. They are believed to have been domesticated by natives of the Andean region of South America over 5000 years ago. The animals are thought to be descendants of a closely related species, *Cavia aperea*, *Cavia fulgida*, or *Cavia tschudii*, and therefore do not exist naturally in the wild.[1] The guinea pig still plays an important role as a food source and in folk medicine and religious ceremonies in this part of the world.[2]

The chinchilla (*Chinchilla laniger*) was introduced to Spaniards in 1524 and named after the Chinchas Indians, a once great nation that had been absorbed into the Inca, who named the animals *Chinchillas*, meaning *little Chinchas*. The demand for chinchilla pelts that followed drove the chinchilla close to extinction and, in 1910, the hunting or exportation of chinchillas was outlawed.[3] According to an apocryphal legend, all of the chinchillas in North America originated from 11 animals—8 males and 3 females—of mixed species (*C laniger* and *Chinchilla brevicaudata*) collected from several different locations and brought to the United States by Mathias F. Chapman in 1923. True or not, the resultant animal is a marvel of physiology. Not only is it the longest living rodent of its size, but it is also remarkably healthy, with only a handful of health problems, most related to poor diet or husbandry.

The aged guinea pig suffers from a long list of diseases and problems. Most important is hypovitaminosis C. Several other problems are directly or indirectly influenced

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by low vitamin C levels, including dental disease and pododermatitis. Dental disease in guinea pigs may be associated with skull size and jaw length as this problem is more prevalent in short-faced animals. Chinchillas have very few old-age disease problems. Both species suffer from renal disease in their last years and many die of complications of kidney failure.

PROBLEMS OF DIET AND HUSBANDRY

Inappropriate diet and husbandry is a major cause of problems for the older guinea pig. Hypovitaminosis C (scurvy) is the most common cause of death in the author's practice and likely contributes to many other health problems of older guinea pigs, including malocclusion, arthritis, and tissue mineralization. Soiled or inappropriate bedding or housing surfaces contribute to pododermatitis, urinary tract infections, and vaginal and scrotal plugs.

Hypovitaminosis C (Scurvy)

Guinea pigs lack the hepatic enzyme 1-gulonolactone oxidase, which is essential for the conversion of glucose to ascorbic acid, a sugar acid with antioxidant properties. The name *ascorbic acid* is derived from *a*, meaning *no*, and *scorbutus*, the medical term for scurvy. Vitamin C is involved in many biochemical processes in the body, including the synthesis of collagen and intracellular ground substance. If vitamin C is not supplemented in the diet, or if the animal is anorexic, scorbutic lesions will rapidly develop.

Clinical signs associated with scurvy vary widely, depending on the level of vitamin C in the diet. Diets containing no vitamin C lead to a more rapidly progressing form of the disease with acutely painful joints and teeth that result in generalized immobility and prevent the consumption of food. The common presentation seen with marginal levels of vitamin C is a chronic form of disease. Signs include frequent vocalizations, weakness, decreased mobility, anorexia, diarrhea, flaky to ulcerative skin lesions, stiffness, petechia of the mucous membranes, subcutaneous hemorrhages, and death due to starvation or secondary infection. Other nonspecific but suspicious signs may include rough hair coat, delayed wound healing, worsening or recurrence of unapparent dermatophyte or scabies infections, changes in patterns of teeth grinding, inactivity, stillbirths, urine scald, and recurrent or chronic disease, including pneumonia and urinary tract infections.[4]

A tentative diagnosis is made based on symptoms and dietary history, while a more definitive diagnosis can be made by analysis of diet, gross and microscopic pathology, and serum ascorbate levels.

Scurvy can be prevented in the guinea pig by supplementing the diet with vitamin C. The daily requirement of ascorbic acid is 15 to 30 mg per guinea pig per day. Pregnant females should receive 30 to 45 mg/d. The author prefers vitamin C supplements added to the drinking water (the author recommends Liquid C [Twin Lab, American Fork, Utah, UT, USA]). Because of instability of vitamin C in the presence of light and chlorine, a solution at a concentration of 200 to 400 mg/L drinking water should be mixed fresh daily. Supplemental feeding of a cup of cabbage, kale, a whole green pepper, or similar foods may be sufficient. However, results are unreliable.

Commercial guinea pig diets are formulated with supplementary vitamin C. The typical feed pellets contain 800 mg/kg at the time of milling. Such factors as dampness, heat, and light can reduce the vitamin C content during storage. Even pellet diets stored under optimal conditions lose their potency within 90 days of milling.[5]

Commercial pet diets may have a shorter storage life—30 to 60 days after milling—and are not a dependable source of vitamin C.

Pododermatitis

Pododermatitis includes a range of lesions of the weight-supporting palmar or plantar surfaces of the feet. Trauma to the foot, caused by wire floors or rough bedding, results in inflammation, atrophy of epithelium, infection, fibrous proliferation, loss of vascularization, necrosis, and then granuloma formation, tendonitis, and osteomyelitis of one or more footpads. Untreated, lesions infiltrate vital structures, resulting in lameness, loss of function, and eventually the death of the animal. *Staphylococcus aureus* is frequently isolated from lesions, entering the skin through cracks from abrasions or exposure to moisture. Predisposing factors include obesity, rough or wire flooring, poor sanitation, and other trauma. In the author's opinion chronic cases may progress to lymphadenopathy, arthritis, tendonitis, and amyloid accumulation in kidneys, liver, adrenal glands, spleen, and pancreatic islets.

Diagnosis of pododermatitis is based on clinical signs. Prognosis is based on a five-level classification taken from that for birds of prey, reported by Remple[6]:

Level I: Lesions involve skin only, no infection or inflammation of underlying tissues, and may be divided into two subclasses: hyperemic and hyperkeratotic.

Level II: Lesions involve skin and the subcutaneous tissues but no gross tissue swelling. Again this level is divided into those with obvious trauma and those with ischemic necrosis of the weight-bearing surface.

Level III: The feet are swollen and painful without obvious involvement of deep tissues. This level is divided into three subclasses based on inflammation: serous, fibrotic, or caseous.

Level IV: Infection extends into deep or vital tissues, but the patient retains normal pedal use of the foot. This is evidence of chronicity. This level may be subdivided once again into two subclasses: fibrotic and caseous lesions.

Level V: Normal pedal function is lost. Infection, inflammation, and tissue destruction has extended into tendon, bone, and joints, resulting in crippling deformities, loss of function, and a grave prognosis.

Medical Treatment

Only the mildest cases of bumblefoot should be treated with medical treatment alone. These would include level I and level II lesions. Healing is slow and lesions may recur or progress. Change bedding to absorbent underpads or sheet liners. Culture lesions and administer appropriate antibiotics. Treat lesions topically with silver sulfadiazine cream (SSD) (Silvadine Cream 1% [Monarch Pharmaceuticals, Inc, Bristol, TN, USA]). Systemic antibiotics, such as azithromycin at 40 mg/kg every 24 hours (Zithromax [Pfizer Inc, New York, NY, USA]) should be administered and modified based on culture results. Treatment must be continued until lesions have healed, which may take weeks to months. Bandaging or a combination of bandages and protective "shoe" dressings may be required for more severe lesions to heal.

Surgery is indicated for level III or greater lesions. Surgery decreases convalescence time in many cases and may improve prognosis. If a single limb is involved with an advanced (level IV or V) lesion, amputation is indicated.

Before surgery, take radiographs to assess bone involvement and prognosis. Osteomyelitis/arthritis adversely affects prognosis for recovery. Treat medically for 2 to 4 weeks before surgery to reduce inflammation and vascularity and to improve long-term prognosis. Buprenorphine and meloxicam are given before induction of

anesthesia and continued for several days postoperatively. Self-adherent bandaging tape (Vetrap Bandaging Tape [3M, St Paul, MN, USA]), cut into a 1-in strip, is wrapped above the elbow/hock as a tourniquet. Wrap just tight enough to take the wrinkles out of the tape to control bleeding without damaging the circulation or nerves of the leg. Clean the feet gently but thoroughly to remove any soil, shedding skin, and debris, and remove any scab.

Using an electrosurgical, radiosurgical, or laser surgical device, make an incision around the lesion. Remove all necrotic tissue and the fibrous capsule of the lesion, and trim skin to full thickness with bleeding edges. Take care to avoid vessels and nerves. Curette to remove any necrotic tissue and irrigate to remove any debris. Close incision in a manner that best retains normal foot architecture and prevents skin from constricting the foot. Use small, 5-0 to 7-0, monofilament absorbable or nylon suture. Place simple interrupted and vertical mattress sutures alternately, beginning and ending with simple interrupted.

Following surgery, bandage the foot using a shoe dressing. A hole is made in the shoe to prevent pressure on the closure and enable visualization of the incision. A variety of materials may be used to create the shoe, including polymethyl methacrylate resin (Jet Denture Repair Acrylic [Lang Dental Manufacturing Co Inc, Wheeling, IL, USA]), thermoplastic casting tape, padded aluminum splint (SAM splint [Jorgensen Laboratories, Loveland, CO, USA]), cotton cast padding, or rubber foam. The shoe should cover the palmar/plantar foot and extend up the posterior lower leg at an angle comfortable for standing.

Postsurgical management includes daily assessment of bandaging and appropriate antibiotics for 2 to 3 weeks. At 2 to 3 weeks, remove shoe bandages and simple continuous sutures. Apply a light-weight bandage over SSD cream (human Band-Aid bandages work well). At 3 to 4 weeks, remove mattress sutures and continue bandaging and SSD for an additional 7 days.

Prognosis is based on the level of the lesion. Level I carries a favorable prognosis with aggressive treatment. Level II and III carry a good prognosis, and level IV lesions have a guarded to poor prognosis. Level IV lesions have a poor to grave prognosis for saving the limb. Consider amputation in vivacious animals. Manage others with analgesics, such as meloxicam at 0.3 mg/kg every 12 hours (Metacam [Boehringer Ingelheim, St Joseph, MO, USA]), and long-term antibiotics therapy.

Spurs

"Spurs" are growths of calluses that form on the lateral side of the carpal foot pad. Some breeds and heavy-bodied animals are more prone to developing spurs, and wire or hard-surface cage flooring appears to contribute as well. These growths may get long, exceeding 0.5 cm, and may contribute to cracks in the carpal pad that develop into pododermatitis. Carefully trim these spurs and file smooth with a fine grit emery board or nail buffer. Reduction of body weight, correction of caging/bedding, and regular application of lotion or softening agent to carpal pads reduce the likelihood of recurrence.

Cheek Tooth Overgrowth and Malocclusion

Chinchillas and guinea pigs have both open-rooted incisors and cheek teeth.[7,8] They are classified as full elodonts.[9] All of the teeth grow continuously, which makes these rodents more susceptible to malocclusion, which in turn leads to conditions that affect the animal's general health.

Guinea pigs and chinchillas grow and wear several inches of teeth annually. If for any reason they are unable to wear their teeth at the rate that they grow, problems develop.

While diet plays a role in tooth wear, the primary force affecting tooth wear is occlusion and bruxing (grinding of the teeth). It is the opposing tooth or teeth that wear rodent teeth. If the excursion of the jaw is limited, or if bruxing is painful, then tooth wear is limited or nonexistent and tooth spurs form or teeth lengthen. If lateral movement of the mandible is not sufficient to cover the opposing cheek teeth, the mandibular cheek teeth wear to form lingual spurs, whereas the maxillary teeth form buccal spurs.[8] Elongated maxillary spurs cause lacerations of the cheeks, while elongated mandibular spurs can form an arch, trapping the tongue. With guinea pigs, this problem of elongated mandibular spurs is often complicated by a collapsing of mandibular cheek teeth associated with loss of periodontal ligaments secondary to scurvy.

Nutrition, genetics, and trauma may play roles in the development of dental pathology. Scurvy, as mentioned above, likely plays a major role in the problem in guinea pigs. Dental pain, resulting from damage to periodontal ligaments and loose teeth, results in the animal not chewing vigorously or long enough to adequately keep up with the rate of growth. The teeth are not worn adequately and overgrow results in tongue entrapment and a mouth that is unable to close. Trauma to teeth caused by external forces, such as falling, or that caused by chewing hard objects, may cause uneven wear and result in overgrowth or formation of spurs.

Clinical signs of cheek-tooth malocclusion include oral pain, anorexia, weight loss, salivation (''slobbers''), ''open mouth,'' incisor malocclusion and overgrowth, facial abscesses, exophthalmos, and ocular discharge. Diagnosis is based on signs, oral examination, and radiographs. Clinicians should obtain well-positioned rostrocaudal and lateral views to examine the occlusal plane and temporomandibular joints, along with right and left oblique views to evaluate for problems of elongated roots, periodontal disease, osteomyelitis, or abscessation. The presence of any of these problems denotes a poorer prognosis.

Treatment should address underlying disease, prevent oral pain, and reestablish normal occlusion. Anesthesia is required for oral examination and the trimming of cheek teeth. Cheek teeth have less enamel than do incisors and may be carefully trimmed with a sharp pair of rongeurs or with a tapered bit on a narrow dental drill. Care must be taken not to damage soft tissues while trimming teeth. For many of these patients, the trimming of their teeth is only a means of treating the symptoms of the underlying problem. For an overwhelming number of these patients, a cure is not possible.

Prognosis in these cases varies with the underlying cause, severity, and duration of the problem. Mild cases of cheek-tooth spurs addressed early have a good prognosis.

Chinchillas are not affected by scurvy, but they do have problems with tooth wear as a complication secondary to having short, brachiocephalic faces. Short-faced chinchillas often have crowding of cheek teeth and resulting impaction, oral pain, elongation of tooth roots, and movement of cheek-tooth location, leading to impaired tooth wear and the formation of dental spurs. Some chinchillas presented with signs of cheek-tooth problems will be found on oral examination to have normal-appearing teeth with well-worn occlusal tables. Radiographs of these animals show dental impaction. Many of these patients will stop salivating and begin eating with the administration of analgesia. ''Rocking'' these patients' cheek teeth with an elevator may help alleviate oral pain. Others appear to ''grow out'' of the problem over a 2- to 6-week period.

Vaginitis and Scrotal Plugs

Wood chips may adhere to the vulva and vestibule, or to the prepuce of guinea pigs, causing a foreign body reaction. Male guinea pigs may accumulate a plug of

sebaceous material, feces, and bedding in the skin fold overlying the interscrotal septum. Treat both conditions by washing the affected area with detergent and water, carefully removing debris. If infected, take cultures and treat the area topically with SSD and administer systemic antibiotics. Problem animals should be placed on a different surface, such as underpads, towels, or waterproof bed pads.

PROBLEMS NOT ASSOCIATED WITH DIET OR HUSBANDRY

Guinea pigs and chinchillas suffer a variety of problems related to metabolism, infection, or just old age. Some of these problems, such as diabetes mellitus and ovarian cysts, may occur in young animals. However, they are more consistently associated with age.

Diabetes Mellitus

Spontaneous diabetes mellitus was first reported in Abyssinian guinea pigs in 1973.[10,11] The disease resembles juvenile diabetes in humans. Studies suggest an infectious cause. About half of a group of females developed diabetes mellitus 6 weeks to 3 months after they were introduced into a colony as breeders.[12] In a group of six guinea pigs, three developed diabetes mellitus over a 12-month period (Jeffrey R. Jenkins, DVM, personal observation, 2001). Other studies showed plaques in embryonated eggs inoculated with urine of affected guinea pigs and found viral particles resembling type C retrovirus.[12]

In a research environment, signs of guinea pig diabetes mellitus most often are seen by 6 months of age. In practice, signs are more commonly seen in mid- to late life. Most often, affected animals show signs of polydipsia, polyuria, and weight loss. Diagnosis is based on abnormal blood glucose levels, glucose tolerance tests, and glucosuria. Affected guinea pigs commonly have urine glucose of greater than 250 mg/dL. Cases treated by the author often have sustained blood glucose levels over 1000 mg/dL. Diabetic guinea pigs do not develop ketoacidosis. Some guinea pigs may have a regression of signs associated with regeneration of beta cells over a period of months to years (unpublished observation).

According to reports, guinea pig diabetes has been successfully treated with insulin. Doses have been based both on glucose levels and normalizing signs of polyuria and polydipsia. The author has used longer acting forms or combinations of long- and short-acting forms. Care must be taken and treatment customized to the patient and the owner's schedule. The author has had a single highly successful case of a guinea pig treated with glipizide at 2 to 5 mg/kg every 12 hours. This animal's owner was highly motivated and followed glucose levels, adjusting the dose as needed. Treatment was continued for over 2 years, at which time glucose levels had returned to normal.

Diabetes mellitus has been reported in chinchillas only three times, and has been linked to obesity. In two cases, animals showed blood glucose levels elevated to at least four times the upper limit of the reference range, as well as severe glycosuria and ketonuria. The animals were treated with insulin and, after initial improvement, both animals died. In the third case, diabetes was at first suspected because of the high blood glucose level and glycosuria, although typical findings, such as ketonuria, polyuria, polydipsia, obesity, and cataracts, were not present. In addition, the serum fructosamine level did not appear to be elevated. The suspected diabetes mellitus in the third case was at first treated with glipizide, 2.5 mg per animal every 12 hours, to stimulate endogenous insulin release (T.M. Donelly, Veterinary Information Network discussion board, May 4, 2009, personal observation).

A single blood glucose concentration measurement is not sufficient for a diagnosis of diabetes mellitus in rodents. Several measurements are required because stress, pain, hyperthermia, and shock can all cause hyperglycemia.

Ovarian Cysts/Rete Cystadenoma

Serous cysts (cystadenoma), originating from cells of the ovarian rete, form in the ovaries of guinea pigs. These cysts have been shown to form in guinea pigs as young as 10 days old and increase in size with age.[13] They are present in all guinea pigs over 1 year of age.[14] In aged guinea pigs, these cysts may be very large. The author has removed cysts measuring greater than 5 cm. Large cysts may affect the function of other organs and the ability to eat a sufficient volume of food.

Clinically, ovarian cysts are associated with signs of depression, anorexia, alopecia, reduced reproductive performance, cystic endometrial hyperplasia, mucometra, and endometritis. As female guinea pigs age, a significant percentage develop a bilateral alopecia. Although no one has proved that the cysts cause the alopecia, the alopecia resolves if an ovariohysterectomy is performed.

Metastatic Calcification

Often asymptomatic, metastatic calcification occurs primarily in mature male guinea pigs.[15] Clinical signs may include weight loss, muscle and joint stiffness, renal failure, or sudden death. Lesions include calcium deposits in the lungs, major organs, the gastrointestinal tract, joints, and skeletal muscles. Organ mineralization is said to be related to diets high in calcium and phosphorus and low in magnesium and potassium. Metastatic calcification is uncommon in guinea pigs fed a diet of hay and green leafy vegetables.

Renal Failure

Perhaps the most common manner in which guinea pigs and chinchillas "die of old age" is renal failure. These patients are typically presented for weight loss, polydipsia/polyuria, or urine scald. Initially laboratory findings include isosthenuria and proteinuria. Only as the disease progresses are blood chemistry values suggestive of renal failure, including elevated blood urea nitrogen and creatinine. In advanced cases, there may be a significant proteinuria, glucosuria, and hematuria. With time, these patients can lose a large portion of their muscle mass and eventually die of primary renal disease or secondary complications.

Trichofolliculoma

Trichofolliculomas are the most common skin tumors of guinea pigs. Two major reviews showed that 45% and 38% of all skin tumors were trichofolliculomas.[16,17] They are benign tumors of the hair follicle epithelium that present as slow-growing cystic masses varying in diameter from 0.5 to 7 cm. They are located predominantly on the back, sides, and lateral thighs. Males are reported to be affected twice as frequently as females. Trichofolliculomas may be found on young guinea pigs, but are most common on animals 3 years and older. Ulcerating tumors and ruptured cysts discharge caseous material. Epidermoid cysts arising from hair follicles are often associated with these tumors, or may arise independently. Treatment is surgical excision.

Other Neoplasms

Guinea pigs and chinchillas have a lower than average frequency of neoplasms.[18]

Guinea pigs

The most common reproductive tumor of guinea pigs is the uterine leiomyoma, which is usually associated with cystic rete ovarii.[19,20] The most common ovarian tumor of guinea pigs is the unilateral teratoma, usually seen in sows over 3 years of age. These benign tumors rarely metastasize. Both male and female guinea pigs can present with mammary tumors, most commonly benign fibroadenomas, but approximately 30% are locally invasive adenocarcinomas that rarely metastasize.[21] The most common respiratory tumor of guinea pigs is bronchogenic papillary adenoma, reported as 30% to 35% of all neoplasms in guinea pigs over 3 years of age. Also, bronchogenic and alveologenic adenocarcinomas have been documented.[22]

Chinchillas

Only a handful of tumors have been reported in chinchillas in the literature. These tumors include lymphoma, uterine leiomyosarcoma, osteosarcoma, and adenocarcinoma of the lung. The author has seen a number of other tumors over the past 25 years. However, there are no notable trends.[23]

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